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Inventor Name	City	State/Country
OLSON, ERIC N.	DALLAS	TEXAS

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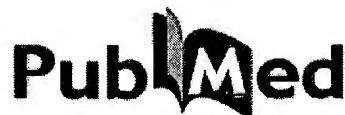
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## Balancing contractility and energy production: the role of myocyte enhancer factor 2 (MEF2) in cardiac hypertrophy.

Czubryt MP, Olson EN.

University of Texas Southwestern Medical Center at Dallas, Dallas, Texas 75390-9148, USA.

Cardiac hypertrophy -- that is, enlargement of the heart resulting from increased myocyte size -- is observed with many forms of human heart disease. It may arise secondary to an insult, such as infarct or chronic hypertension, or may occur as a consequence of a genetic defect, such as in hypertrophic cardiomyopathy. Traditionally, it has been widely believed that hypertrophy occurred as an adaptive response to normalize increased wall stress due to disease. Recently, however, it has been observed that while hypertrophy initially appears to improve the function of the heart following insult, over time, it frequently leads to a decompensated state, characterized by fibrosis and chamber dilation, resulting in overt heart failure. Hypertrophy also occurs during fetal development, immediately after birth, and in trained athletes; however, it does not lead to decompensation in these states. Experiments over the last 15 years have implicated similar signaling pathways in both pathological and physiological hypertrophic responses. Recently, important differences have been demonstrated that might hold the key to the development of effective new treatments for human diseases. This chapter focuses on how these hypertrophic responses differ from one another phenotypically and discusses how inefficient or impaired energy metabolism in the heart may contribute to the development of pathological responses. We also discuss recent evidence that the myocyte enhancer factor 2 (MEF2) transcription factor family, which previously has been shown to be important in cardiac development and hypertrophy, may have a role in regulation of cardiac energy metabolism.

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